

Acute stress impairs children's sustained attention with increased vulnerability for children of mothers reporting higher parenting stress

Leslie E. Roos  | Ryan J. Giuliano | Kathryn G. Beauchamp | Elliot T. Berkman  | Erik L. Knight | Philip A. Fisher

Department of Psychology, University of Oregon, Eugene, Oregon

Correspondence

Leslie E. Roos, Department of Psychology, Duff Roblin Building, 190 Dysart Road, University of Manitoba, R3T 2N2.
Email: Leslie.roos@umanitoba.ca

Present address

Leslie E. Roos and Ryan J. Giuliano, Department of Psychology, University of Manitoba, Winnipeg, MB, Canada

Kathryn G. Beauchamp, Department of Psychiatry, University of New Mexico, Albuquerque, New Mexico

Erik L. Knight, Center for Healthy Aging, The Pennsylvania State University, State College, Pennsylvania

Funding information

This work was supported by grants AG048840, CA175241, and DA035763 from the National Institutes of Health and grant CA1818-02-01 from the Administration for Children and Families.

Abstract

Despite evidence that acute stress impairs attention in adults, there has been minimal research in children. Here, the effects of acute stress on Go/No-go performance were examined in young children (M age = 5.41 years). Given the critical role of the parent-child relationship to children's self-regulatory development, the extent to which parenting stress predicts children's cognitive vulnerability to acute stress and autonomic reactivity was also investigated. A between-groups design ($n = 58$ stress, $n = 26$ control) was used with oversampling of the stressor-exposed children to examine individual differences. The Parenting Stress Index and subscales were employed as a measure of parenting stress. Acute stress impaired children's sustained attention, but not inhibitory control. Higher parenting stress was associated with vulnerability to attentional impairment. Parenting distress was also positively associated with sympathetic reactivity to acute stress, but neither sympathetic nor parasympathetic reactivity was associated with attentional impairment. A conceptual model of pathways through which repetitive acute stress may contribute to self-regulatory difficulties is presented, including the potential buffering role of caregivers.

KEYWORDS

acute stress, parenting stress, sustained attention

1 | INTRODUCTION

A substantial body of research has delineated links between chronic stress exposure and childhood deficits in cognitive function (Crowell, Cicchetti, Rogosch, & Toth, 2015; Fisher et al., 2016; Pechtel & Pizzgalli, 2011). This work has been important for highlighting the relevance of the early environment to cognitive development and identifying repetitive/chronic stress as a pathway through which experience imprints on cognition. Missing from this literature, however, is an examination of the impact of temporally proximal acute stressors on cognitive function, which may be relevant for understanding long-term deficits observed in individuals

with repetitive stress exposure. The identification of contexts that induce cognitive vulnerabilities following acute stress has particular importance due to the developmental plasticity of biological systems supporting both stress management and effortful cognitive function during childhood (Berkman, Graham, & Fisher, 2012; McEwen & Morrison, 2013).

In adults, acute stress has been shown to impair attention (Olver, Pinney, Maruff & Norman, 2015; Scholz et al., 2009; Vinski & Watter, 2013) and inhibitory control processes (Roos, Knight, et al., 2017). Theory suggests that acute stress biases cognitive function toward more indiscriminately reactive states, with neuroimaging research highlighting alterations in early attentional processes, even

after a stressor has ended (Sänger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014; van Marle, Hermans, Qin, & Fernández, 2009). Although the allocation of resources toward cognitive processes that enable vigilance toward potential threats is theorized to have protective benefits *under* stress, it can result in ongoing “costs” to other top-down goal-directed behaviors if such vigilance is maintained after a stressor has passed (Sänger et al., 2014).

A candidate factor that may influence individual differences in children's cognitive vulnerability to acute stress is the parental context to which they are exposed. The parent-child relationship is critical to supporting children's ability to both manage acute stress (Bridgett, Burt, Edwards, & Deater-Deckard, 2015; Gunnar, Hostinar, Sanchez, Tottenham, & Sullivan, 2015) and develop effortful cognitive skills (Fay-Stammach, Hawes, & Meredith, 2014). As delineated below, stress relevant to the parent-child relationship (i.e., parenting stress) could be theorized to impact multiple aspects of children's ability to recover from acute stress, including low parental responsivity to child distress, limited parental scaffolding of stress-management skills, or parental modeling of high distress in response to challenging situations. Should parenting stress be linked to acute stress vulnerability, it would also represent a promising intervention target for reducing children's cognitive vulnerability.

Similarly, the extent to which individuals differentially engage their autonomic nervous system (ANS), comprised of the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS), in response to stress may influence vulnerability to the cognitive effects of acute stress given the importance of the ANS in regulating arousal and resource allocation. Specifically, the SNS facilitates heightened arousal and resource mobilization (e.g., increased heart rate, blood flow, and metabolic mobilization; Gunnar & Quevedo, 2007), whereas the PNS allows for rapid heart rate changes to meet fluctuating contextual needs through top-down brainstem connections via the vagus nerve (Berntson, Cacioppo, & Quigley, 1993; Porges, 2007).

Here, we employ a between-groups experimental design to examine if exposure to an acute stressor alters preschool-aged children's sustained attention and inhibitory control, as indexed by performance on a Go/No-go task. Within laboratory stress-exposed children, we characterize the extent to which individual differences in parenting stress are predictive of children's post-stressor cognitive impairment. Finally, we examine the extent to which parenting stress is associated with alterations in children's autonomic system reactivity and whether such alterations are associated with children's cognitive vulnerability to the effects of acute stress. The rationale for each key hypothesis is delineated, below.

Hypothesis 1: *Acute stress impairs cognitive performance in young children.*

Although the impact of acute stress on cognitive performance has not been previously characterized in children, a small number of adult and adolescent studies suggest that both sustained attention and inhibitory control can be affected by laboratory induced stress.

It is theorized that, following acute stress, cognitive function remains biased toward the indiscriminant “bottom-up” processing of environmental stimuli leading to difficulty in maintaining performance on effortful attention tasks (Sänger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014; van Marle et al., 2009).

Research using both Go/No-go tasks (Scholz et al., 2009; Vinski & Watter, 2013) and other measures of attention (i.e., change detection, sustained attention to response; Alomari, Fernandez, Banks, Acosta, & Tatar, 2015; Sänger et al., 2014) provides evidence for impaired sustained attention performance, as indexed by slower and/or greater variability in reaction time. However, when sustained attention is impaired, the validity of no-go accuracy as an index of inhibitory control per se is compromised because strong attentional engagement is a prerequisite for accurate performance on no-go trials in a Go/No-Go task. It is not surprising, then, that several studies have shown null results on no-go accuracy in the presence of lower attentional engagement (e.g., Band, Ridderinkhof, & van der Molen, 2003). In contrast, adult research using the stop-signal task, which explicitly adjusts for the speed of responses on Go trials when estimating inhibitory control performance, has found that acute stress does impair inhibitory control (Roos, Knight, et al., 2017). Based on these findings in adults, it was expected that acute stress would impair sustained attention performance in children. We did not have directional hypotheses about the presence of the effect of stress on inhibitory control (i.e., No-go performance), given mixed evidence for impairment in adults.

Hypothesis 2: *Parenting stress impairs children's ability to manage stress.*

We hypothesized that parenting stress would be associated with children's vulnerability to acute stress, given the critical role of the parent-child relationship to supporting children's ability to both manage acute stress and develop effortful cognitive skills (Bridgett et al., 2015; Fay-Stammach, Hawes, & Meredith, 2014; Gunnar et al., 2015).

By providing warm and responsive caregiving to children's distress, parents help children regulate their physiology and emotional state (Haley & Stansbury, 2003; Liu et al., 1997). With diminished caregiver support and lower quality parent-child relationships, children experience less maternal buffering during acutely stressful experiences (Hostinar, Sullivan, & Gunnar, 2014). Over time, parents' supportive responding to children's negative emotional experiences is believed to give children autonomy in managing their own emotions and achieving resiliency from stressful experiences (Bridgett et al., 2015; Sanders, Zeman, Poon, & Miller, 2015). Social learning is also likely involved in stress regulation, as children learn to model parent's stress-management techniques (Bridgett et al., 2015; Cummings, Iannotti, & Zahn-Waxler, 1985; Moed et al., 2016). Multiple studies have found that higher parental distress to negative emotions and poor bonding (i.e., low attachment quality) are both associated with children's elevated SNS reactivity to acute stress (Monti, Abaied, Rudolph, 2014; Oosterman, De Schipper, Fisher,

Dozier, & Schuengel, 2010). Research in infants finds that greater maternal sensitivity is linked to less PNS reactivity to separation events (Moore et al., 2009; Propper et al., 2008), suggesting that parenting may buffer infants' physiological experience of distress.

The preschool years are an important developmental window for the emergence of children's independent regulatory capacities (Tan Camras, Deng, Zhang, & Lu, 2012; Zajicek-Farber, Mayer, & Daugherty, 2012). Prior to this time, children are reliant on their caregivers to help them navigate challenging situations, with children's displays of distress signaling their need for physical comfort or emotional support (Bridgett et al., 2015). However, as children become more independent, they increasingly encounter stressful situations separate from primary caregivers in contexts that require them to regulate their arousal, attention, and behavior to effectively navigate and recover from a given situation. High levels of parenting stress may influence multiple aspects of children's ability to recover from acute stress, including low parental responsiveness to child distress or limited parental scaffolding of stress-management skills.

Here, we employed the Parenting Stress Index (PSI, Abidin, 1995; Reitman, Currier, & Stickle, 2002) which quantifies stress by sub-components of parental distress (sense of competence, social support, depression), parent-child dysfunctional interactions (bonding, rejection, unsatisfying interactions), and difficult-child temperament (oppositonality, challenges of effective behavior management). We expected that children's experiences of being raised by a mother with higher parenting stress would be linked to lower capacity to cope with acute stress resulting in a challenge in re-engaging in an effortful laboratory task. Accordingly, exposure to mothers with higher parenting stress was expected to be associated with children's greater impairment in effortful cognitive function (i.e., Go/No-Go task performance) immediately following acute stress. Because of the limited research to date on how different aspects of children's rearing environments may contribute to acute stress vulnerability, exploratory PSI subscale analyses were conducted to guide future research.

Hypothesis 3: *ANS acute stress reactivity will predict greater cognitive vulnerability.*

A final question was whether individual differences in children's ANS reactivity to acute stress would be associated with post-stress cognitive impairment and mediate links between parenting stress and cognitive impairment. In particular, we were interested to examine whether ANS reactivity reflects physiological coping styles that increase children's cognitive vulnerability to the effects of acute stress, given previous research suggesting that PNS activity may buffer adult cognitive function from acute stress (Roos, Knight, et al., 2017). However, this question was largely exploratory given the lack of prior research in this domain in young childhood.

The acute stressor employed in this study affords the unique opportunity to test theories about the role of ANS activity and concurrent cognitive function under conditions where PNS and SNS levels are manipulated in a targeted direction to facilitate rapid responding

under social threat (SNS engagement, PNS disengagement; Roos, Giuliano, et al., 2017). The role of SNS reactivity in supporting cognitive performance is debated, with some theories suggesting that engagement reflects the appropriate allocation of physiological resources, whereas other theories suggest that engagement may reflect a perception of low baseline resources and high resource mobilization needs resulting in an over-aroused state (reviewed in Mendes & Park, 2014). Evidence suggests that greater SNS engagement to a challenge task predicted better memory of the task (Quas, Carrick, Alkon, Goldstein, & Boyce, 2006) and higher task level SNS predicted better auditory selective attention (Giuliano et al., 2018). PNS reactivity has also been theorized to relate to cognitive function given that shared prefrontal brain regions are linked to the regulation of both attentional resources and limbic system activity (Graziano & Derefinco, 2013; Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). However, findings in childhood have been mixed. Some studies find that moderate levels of vagal withdrawal are linked to higher cognitive performance (e.g., executive function battery; Marcovitch et al., 2010) and other research failed to document associations between PNS reactivity and cognitive performance (i.e., intelligence scores; Staton, El-Sheikh, & Buckhalt, 2009). Notably, prior child research has not characterized links between SNS or PNS to acute stress and *subsequent* cognitive impairment.

1.1 | The present study

In this study, we examined the effects of acute stress on Go/No-go performance in a sample of young children. We hypothesized that laboratory-induced stress would impair children's performance, particularly sustained attention. Notably, because this investigation was conducted in the young childhood age range, characterized by a lack of ceiling effects on Go trial accuracy and highly variable response times, our outcome measures of interest were performance accuracy, consistent with other research in this age range (e.g., Loman et al., 2013; Palmer, Miller, & Robinson, 2013; Yu, Kam, & Lee, 2016). Accordingly, errors of omission on Go trials reflect sustained attention failures, whereas errors of commission on NoGo trials reflect inhibitory control failures (Palmer et al., 2013). We oversampled the acute stress group relative to controls, in order to conduct follow-up analyses of individual differences in stress-exposed participants.

Individual differences in parenting stress were examined as a predictor of differential vulnerability to the effects of acute stress. It was hypothesized that, among stressor-exposed children, overall parenting stress would exacerbate children's cognitive impairment from the acute stressor. We also explored the extent to which cognitive impairment would be predicted by the subscales of parenting stress. Links between cognitive impairment and the "higher parent distress" and "parent-child dysfunctional interactions" subscales might be consistent with theories about the intergenerational transmission of stress regulation (e.g., skill scaffolding, social learning) and associations with the "difficult child" subscale could be conceptualized as a more temperamental risk factor for cognitive vulnerability to acute stress.

Finally, we characterized the extent to which biological measures of acute stress responsivity were linked to differences in parenting stress and vulnerability to effects of acute stress. It was hypothesized that greater parenting stress would predict greater SNS reactivity to acute stress, which would mediate associations between parenting stress and cognitive impairment. Examination of associations between parental stress and RSA were largely exploratory given the limited previous work in this area. Establishing the extent to and mechanisms by which acute stress alters children's subsequent cognitive performance may allow us to understand the contexts in which children are more likely to have cognitive difficulties and identify candidate processes, such as acute stress management, for targeted intervention research that may increase children's cognitive function.

2 | METHODS

2.1 | Participants

Eighty-four mother-child dyads volunteered to participate through community recruitment. Children (age, $M = 5.38$ years, $SD = 0.65$, range = 4.20 to 6.71 years) were randomly assigned to a Control ($N = 26$, 14 female) or Stressor condition ($N = 58$, 33 female), with more children in the Stressor condition to permit examination of within-condition individual differences. Pre-enrollment screening excluded children with a history of psychiatric disorders, developmental delays, or serious health problems. Mothers reported a range of household incomes (median = \$25,000–\$29,999; range < \$4,999–\$100,000+) and education (median = some college or associate's degree; range: less than high school, to graduate or professional degree), with no significant differences in sociodemographic variables (child age, child sex, household income, maternal education) between stressor and control conditions (all $ps > .05$).

2.2 | Procedure

Participants completed one 2-hr laboratory visit during which all measures were collected. Start times ranged from 9 a.m. to 3 p.m., with children awakening at least 1 hr prior and not eating 1 hr prior to the visit. Mother and child dyads arrived at the laboratory and were greeted by two assessors who escorted them to a confidential laboratory testing area. One assessor focused on engaging the child with their role as a "Zookeeper Explorer" during the laboratory visit and animal-related activities, including a coloring book and sticker chart describing the activities to be completed during the visit. Following a description of laboratory activities, written consent was obtained from the mother and verbal assent from the child. Next, the mother assisted the assessors in encouraging child comfort with the application of electrodes for monitoring of ANS physiology and an EEG net (EEG measures not reported here).

Testing began approximately 45 min after laboratory entry, with mother and child sitting in chairs across a table while facing each other but not making physical contact, and watching a 5-min

peaceful ocean video to assess baseline ANS activity (Piferi, Kline, Younger, & Lawler, 2000). After the baseline ANS collection, the mother left the room to complete questionnaires, and the child completed a "Pre" Go/No-go task, followed by a matching task (either stressor or control condition), and then a "Post" Go/No-go task. Following the post Go/No-go task, mother and child were reunited and additional laboratory activities were conducted (not described here). ANS measures were collected throughout the matching task to examine effects of reactivity relative to the initial baseline assessment. Notably, with the exception of the matching task, all laboratory procedures for the stressor and control condition were identical regarding assessments and children's interactions with their mothers and assessors.

2.3 | Measures

2.3.1 | Go/No-go task

Children performed a child-friendly version of a zoo-themed Go/No-go task with positive or negative feedback on No-go trials (Grabell et al., 2017; Grammer, Carrasco, Gehring, & Morrison, 2014). First, children were told a short story about animals escaping a zoo. Next, children were told that their job was to press a button as quickly as possible to catch the animals, each time an animal picture (Go stimuli) was presented on the screen. However, if a chimpanzee picture was presented (No-go stimuli), they were instructed to withhold a button press because the chimpanzees were helping to catch the other animals. Research assistants emphasized the importance of going quickly and praised the children for catching animals in order to increase the prepotency of the Go response. Children completed three practice blocks to ensure that they understood task instructions: (a) self-paced Go-only trials, (b) speeded response with Go-only trials, and (c) speeded response with mixed Go and No-go trials.

The task included two blocks of 60 trials each, with No-go stimuli presented 25% of the time. Children completed two task blocks pre- and post-stressor. During each trial, animal stimuli were presented for 1,200 ms, followed by a fixation cross for 1,500 ms or until a response was made. The intertrial interval consisted of presentation of a fixation cross for 500 ms. On Go trials, when children responded correctly, they proceeded to the next trial; however, if they failed to respond quickly enough, an unhappy face was presented to indicate trial failure. Children were told that these unhappy faces indicated a need to respond faster on Go trials. Happy faces were presented for each correct No-go trial, and unhappy faces were presented for each incorrect trial.

Animal stimuli were adapted from previous Go/No-go task versions in order to have two distinct sets of animal stimuli (forest- and savanna-themed) for use pre- and post-stressor. The forest and savanna picture sets were presented in a counter-balanced order across subjects. Animal stimuli were all vertically cropped images emphasizing the animal's face, in an attempt to maximize ease of identifying the animal and consistency of stimuli, as well as to

minimize horizontal eye movement. The forest set consisted of pictures of bears, deer, foxes, raccoons, wolves, and cougars, whereas the savanna set consisted of pictures of giraffes, lions, meerkats, zebras, and dingos.

Consistent with previous research in similar-age samples, inclusionary criteria required a baseline (i.e., pre-stressor) Go trial accuracy of >50% (Cragg, Fox, Nation, Reid, & Anderson, 2009; Roos, Pears, Bruce, Kim, & Fisher, 2015). This cut-off was created to ensure that children were willing and able to participate in the task, resulting in the exclusion of seven children pre-stressor. An additional three children refused to participate in the task post-stressor, resulting in a total of 74 children with complete behavioral data (49 Stressor, 25 Control). Missingness was not associated with sociodemographic covariates (assessed by bivariate correlations, all $ps > .05$). Accuracy descriptives for the Go/No-go task are reported as percent correct in Table 1. All results were consistent with or without excluded participants.

2.4 | Acute stressor manipulation

The stressor employed in this study was a matching task adapted from previous research (Kryski, Smith, Sheikh, Singh, & Hayden, 2011; Tolep & Dougherty, 2014) and described in detail elsewhere (Roos, Giuliano, et al., 2017). Briefly, across stressor and control conditions, children played a matching game in which they were asked to use a color-coded legend to match colored stickers to transportation types on a worksheet with 30 squares to complete. Children performed this task on three consecutive 2-min trials, but were not able to complete the task, because 2 min is an insufficient time for most 4- to 6-year-old children to complete the worksheet. Two children were close to finishing the worksheet within 2 min, so the task was covertly stopped ~10 s short and the stop light flashed, so that these participants experienced task failure.

In the stressor condition, children picked a desired prize to win for successfully completing a worksheet. The assessor, an unfamiliar adult who used stern, flat affect, operated a stoplight that was set to green for 90 s, then yellow for 30 s, and immediately afterward to red, accompanied with a loud beep signaling the end of a trial. The assessor provided negative feedback twice each trial (e.g., "You need to go faster.") and after three trial failures, children were told they did not earn their prize. Next, the assessor left the room and a

friendly, familiar assessor returned. Following the end of the experiment (i.e., after the post-stress Go/ NoGo), the child was debriefed about the nature of the task and was told that they did an excellent job and earned their desired prize.

In the control condition, facilitated by a friendly, familiar assessor, the children were told that after two minutes it was time to work on the next worksheet. There was no mention of prizes, winning, or losing.

2.5 | Children's biological acute stress responsivity

Regarding autonomic physiology, a montage of eleven electrodes was used for the measurement of respiratory sinus arrhythmia (RSA) and pre-ejection period (PEP), indexing PNS and SNS activity, respectively. Electrocardiogram (ECG) was obtained from three disposable pre-gelled electrodes placed in a modified Lead II setup on the distal right clavicle, lower left rib, and lower right abdomen. Cardiovascular impedance (Z0) was recorded from eight electrodes placed in a tetrapolar configuration on the left and right lateral neck and torso, from a vertical maximum of the jawline down to the diaphragm. Data were acquired wirelessly via Biopac Nomadix BN-RSPEC and BN-NICO transmitters (Biopac Systems Inc) sending ECG and impedance signals, respectively, to a Biopac MP-150 acquisition unit placed in the room with the participant. A respiration signal was derived from the raw impedance cardiogram for the inspection of respiration values. Consistent with other studies of the age range reported here, RSA values were derived from natural log transformed values of the spectral power in the high-frequency range (0.24–1.04 Hz) of the ECG signal (Calkins & Keane, 2004; Roos, Knight, et al., 2017). PEP was calculated from the first-order derivative of the cardiovascular impedance signal (dZ/dt), as the length of time from the Q-point of the ECG waveform to the B-point of the dZ/dt waveform (Berntson, Lozano, Chen, & Cacioppo, 2004).

Mindware HRV and IMP softwares were used to process physiological data. Confirmation of heart beats was performed via visual inspection of ECG signals in 30-s epochs. Edited ECG files were then used for the processing of PEP values, whereby visual inspection was performed to verify that both the Q- and B- points were present and correctly placed in 30-s averages of ECG and dZ/dt waveforms. For RSA and PEP, epochs were averaged across baseline, first-half matching task, and second-half matching task. Due to exaggeration of the stress-inducing qualities (i.e., repeated failure) in the second-half of the matching task, the first-half and second-half of the matching task were examined separately. Among participants with usable behavioral data, exclusion due to physiological data resulted from refusal of electrodes ($N = 4$) and less than 50% artifact-free data for baseline measurement or second-half of the matching task (RSA, $n = 8$; PEP, $n = 17$). Therefore, analyses included 68 participants (42 Stressor; 20 Control) with RSA data and 53 participants (40 Stressor, 13 Control) with PEP data.

As previously reported, between group repeated measures ANOVAs confirmed that the acute stressor elicited significant responsivity across systems compared to the control condition,

TABLE 1 Go/No-go accuracy performance descriptives

	Stressor N = 49 M(SD)	Control N = 25 M(SD)
Go pre accuracy	0.83 (0.13)	0.81 (0.16)
Go post accuracy	0.80 (0.17)	0.82 (0.17)
Go post-pre accuracy change	-0.04 (0.11)	0.00 (0.09)
No-go pre accuracy	0.72 (0.15)	0.76 (0.12)
No-go post accuracy	0.72 (0.14)	0.71 (0.18)
No-go post-pre accuracy change	0.01 (0.12)	-0.05 (0.13)

including greater RSA withdrawal and greater shortening of PEP (Roos, Giuliano, et al., 2017). Paired sample *t* tests within the stressor group further characterized the reactivity, with significant changes from second half of the matching task for RSA and PEP. The average baseline value for RSA was 6.58 ($SD = 1.12$) and for PEP was 90.66 ms ($SD = 10.22$). The average reactivity for RSA was -1.06 ($SD=0.75$) and for PEP was -3.17 ($SD = 9.21$). Prior research has also documented the presence of cortisol reactivity among the stressor exposed participants, which was absent in the control condition, suggesting that the employed task was an effective laboratory paradigm for inducing acute stress across systems (Roos, Giuliano, et al., 2017).

2.6 | Parenting stress index

The Parenting Stress Index—Short Form (PSI-SF) includes 36 items designed to assess parenting stress (Abidin, 1995). This version was created based on factor analysis of the subscales that best loaded together from a larger 120 item Parenting Stress Index survey and has been validated with sociodemographically diverse samples (Abidin, 1990; Reitman, et al., 2002). The PSI-SF includes three subscales, each consisting of 12 items, indexing parental distress, parent-child dysfunctional interaction stress, and stress related to having a difficult child. Higher scores reflect higher stress. The parental distress subscale measures parent's perception of their own distress related to aspects of child-rearing, spousal relationships, social support, and other life roles. It includes questions such as *I feel trapped by my responsibilities as a parent* and *I feel that I cannot handle things*. The parent-child dysfunctional interactions subscale seeks to measure stress related to negative interactional styles between parent and child with questions such as *My child rarely does things that make me feel good*. In contrast, the difficult child subscale measures difficult child temperament (i.e., defiance, noncompliance, and demandingness) and includes questions such as *My child is moody and easily upset*. These scales have been found to have very high internal consistency across the three subscales and the total stress scale (e.g., Abidin, 1995), which were replicated in this study (Chronbach's $\alpha = .80-.91$). Also consistent with validation studies are the high correlations between subscales and the total scale score (Pearson correlation $r_s = .52$ to $.87$, all $p_s < .001$ in the present sample).

If a respondent refused or missed an individual item within a subscale, their scale and total scores were estimated as the mean of existing scale items multiplied by the total number of scale questions. In this study, eight respondents were missing a single item and two respondents declined to respond to any questions. The sample means for the subscales were as follows: parental distress $M(SD) = 25.93(7.45)$; parent-child dysfunctional interaction $M(SD) = 18.05(5.79)$; difficult child $M(SD) = 26.98(6.62)$; total stress $M(SD) = 70.96(16.49)$. We note that these group average scores are similar to previous research conducted in non-clinical samples including Head Start populations and families with children transitioning into kindergarten (DeCaro & Worthman, 2011; Reitman et al., 2002).

2.7 | Analytic plan

All analyses were conducted in SPSS (Version 22.0). Preliminary analyses were conducted to determine if sociodemographic covariates (child age, child gender, household income, caregiver education) predicted change in Go or No-go performance measures for inclusion in multivariate analyses. A repeated measures ANOVA (condition \times trial-type \times time) was then used to examine the effects of condition (stressor, control) on performance by trial type (Go, No-go), across time (pre-stressor, post-stressor). Within the ANOVA, simple effects of time (pre-to-post) within condition were used to examine changes in performance for Go and No-go trial types by condition (Berkman & Reise, 2011). Next, within the stressor group, we aimed to characterize the extent to which parenting stress was predictive of individual differences in performance impairment, as calculated as accuracy change from pre to post stressor. One participant's change score was Winsorized for individual difference analyses as it was $>3SD$ above the mean. Finally, we examined the extent to which SNS and PNS reactivity were linked to parenting stress and cognitive vulnerability to acute stress.

3 | RESULTS

Preliminary covariate analyses found no associations between sociodemographic covariates and Go or No-go performance change measures and were not included in subsequent analyses. Because sociodemographics also did not differ between groups, correlational analyses were determined to be appropriate to examine individual differences among stressor-exposed participants for Hypotheses 2 and 3.

Hypothesis 1: *Acute stress impairs cognitive performance in young children.*

Results of a repeated measures ANOVA (condition \times trial type \times time) predicting behavioral performance demonstrated significant main effects of trial type [$F(1,72) = 19.75, p < .001, \eta^2 = 0.22$], with higher Go relative to No-go trial accuracy. Of particular relevance to the main hypotheses, we observed a significant interaction of time \times trial type \times condition [$F(1,72) = 4.17, p = .045, \eta^2 = 0.06$]. Planned follow-up simple effects investigating change in performance per condition revealed a decrease in Go trial performance for the stressor group [$F(1,72) = 4.47, p = .038, \eta^2 = 0.06$] and no change in Go trial performance for the control group [$F(1,72) = 0.01, p = .910$] (see Figure 1). There were no significant changes in No-go performance for either group; stressor [$F(1,72) = 0.11, p = .743$], control [$F(1,72) = 3.39, p = .070$]. Full descriptive results of condition \times trial type \times time are presented in Table 1.

Hypothesis 2: *Parenting stress impairs children's ability to manage stress.*

Individual difference analyses within the stressor group indicated that greater scores on parenting stress overall was associated

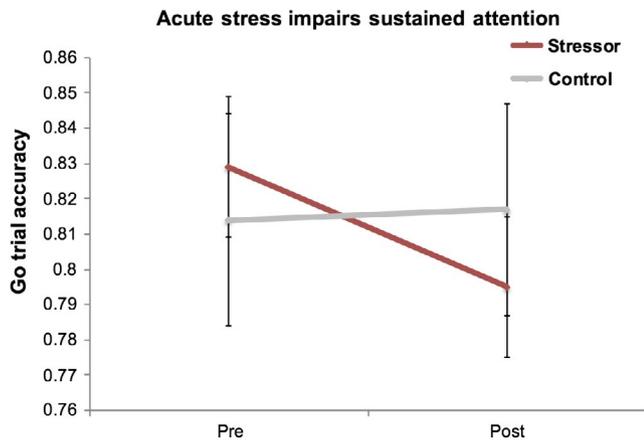


FIGURE 1 Simple effects of acute stress, versus control, and go performance

with Go performance decline [$r(47) = -.34, p = .02$; Table 2; Figure 2]. Subscale analyses of the parenting stress short-form scale indicated that Go performance decline was significantly related to the parent distress [$r(49) = -.32, p = .03$] and mother-child dysfunctional interaction [$r(41) = -.30, p = .04$] subscales, but not the difficult child subscale [$r(41) = -.25, p = .09$; Table 2]. We note that a correlational approach was prioritized due to the complexity in interpreting four-way interactions (time \times trial type \times condition \times parenting stress) as well as the limited statistical power and lack of apriori hypotheses in the control condition. However, we did run an ANOVA to ensure that we did not miss any insights from this approach and confirmed that results were consistent with the more straightforward correlational analyses.

Hypothesis 3: *ANS acute stress reactivity will predict greater cognitive vulnerability.*

Finally, we tested the extent to which stressor-exposed children's RSA and PEP reactivity were associated with overall parenting stress and parenting stress subscale measures, as well as with Go performance vulnerability. Neither autonomic measure was

associated with Go performance decline (Table 2). PEP reactivity was associated with the parental distress subscale of the parenting stress measure ($r(38) = -.45, p = .004$) such that greater parent distress predicted increased SNS activation to the stressor (Figure 3), but no other correlations between PSI total or subscale measures were associated with ANS reactivity measures (Table 2). Baseline ANS function was not associated with Go performance decline (all $ps > .05$). Because there were no associations between ANS reactivity (or baseline measures) and Go performance decline, subsequent mediation models (i.e., ANS reactivity mediating links between parenting stress and performance impairment) were not examined.

4 | DISCUSSION

In a sample of healthy young children, we demonstrated that an acute stressor significantly impaired cognitive performance on a child-adapted Go/No-go task relative to a control condition. Specifically, children exposed to acute stress performed significantly worse on a measure of sustained attention (Go trials) at post-relative to pre-stressor, with no differences in inhibitory control performance (No-go trials). Children of mothers who self-reported greater parenting stress had elevated vulnerability to the effects of acute stress on sustained attention. Given that the age range (~4.5–6.5 years) studied here is an important period for children to gain more autonomous distress tolerance skills, these findings may have implications for children's ability to engage in goal-directed behavior involving attention following acutely stressful events.

Although a growing body of research in adults has sought to characterize the effects of acute stress on cognitive performance (Roos, Knight, et al., 2017; Scholz et al., 2009; Vinski & Watter, 2013), similar research has not been conducted in children. The results of this study extend research in adults identifying post-stressor impairment in sustained attention performance (Alomari, Fernandez, Banks, Acosta, & Tartar, 2015; Sanger et al., 2014; Scholz et al., 2009; Vinski & Watter, 2013). Because this study was focused on a young childhood sample, our outcome measures of interest were

	1.	2.	3.	4.	5.	6.	7.
1. Go post-pre accuracy change	—						
2. PSI total	-0.34*	—					
3. PSI maternal distress	-0.32*	0.88***	—				
4. PSI dysfunctional interactions	-0.30*	0.83***	0.62***	—			
5. PSI difficult child	-0.25	0.83***	0.58***	0.54***	—		
6. PEP reactivity	-0.06	-0.29	-0.45**	-0.20	-0.05	—	
7. RSA reactivity	-0.09	0.07	-0.04	0.11	0.12	0.39*	—

TABLE 2 Maternal stress and biological reactivity associations with Go performance vulnerability amongst stress-exposed participants

* $p < .05$.

** $p < .01$.

*** $p < .001$.

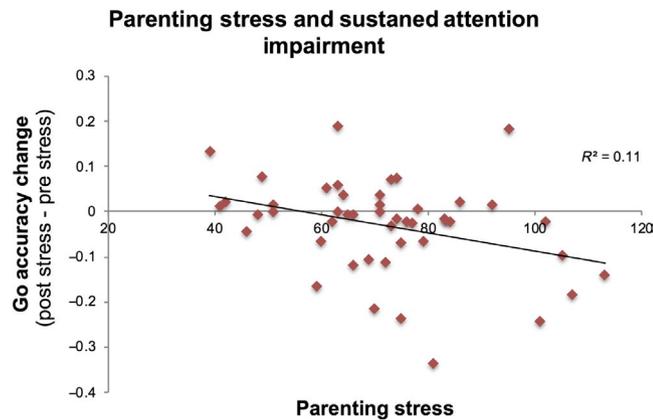


FIGURE 2 Maternal stress predicts stressor-induced sustained attention impairment

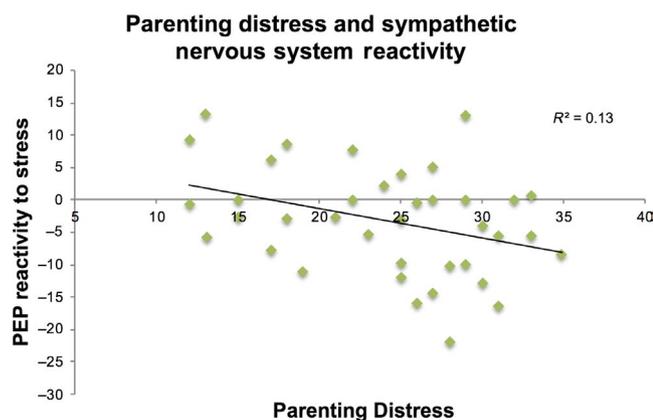


FIGURE 3 Maternal distress predicts PEP shortening in response to acute stress

performance accuracy, as opposed to measures of reaction time used in adult samples, consistent with other research in the young childhood age range (e.g., Yu et al., 2016). Given established associations between impaired cognitive performance and exposure to both chronic (i.e., across years, Cowell, et al., 2015) and recent (i.e., within days; McCoy, Raver, & Sharkey, 2015) stressors, we suggest that a consideration of the effects of acute stressors on EF may be of value for further narrowing in on contexts in which stress affects children's cognitive function.

The present results indicate that acute stress impairs children's ability to perform on sustained attention trials of the Go/No-go task, resulting in more errors of omission on Go trials. Research in adults suggests that impaired sustained attention following acute stress is linked to more self-reported mind-wandering and diminished attentional resources as indexed by pupil dilation (Vinski & Watter, 2013). Alternatively, children may have heightened sensitivity to No-go stimuli and the possibility of making errors post-stressor, and therefore take a highly cautious response strategy in which they prioritize avoiding errors of commission so that they are not able, or willing, to make a response within the Go trial response window. It is also possible that the diminished sustained attention performance

is linked to disengagement from the task or lower motivation following acute stress. Future research could systematically investigate techniques to improve children's motivation, such as stickers or food rewards, and examine the extent to which it improves performance for stress-exposed children.

The finding that acute stress did not impair children's No-go inhibitory control performance is consistent with previous adult research using the Go/No-go task. But these results are inconsistent with other research using the stop signal task, which found evidence for acute stress induced inhibitory control impairment (Roos, Knight, et al., 2017; Scholz et al., 2009). Given the competing demands of Going and Not-going in a Go/No-go task, it is challenging to determine the extent to which an acute stressor affects children's inhibitory control, if the prepotent response is diminished due to reduced Go trial responding. Because a single cue (i.e., an animal image appearing on the screen) indicates both "go" and "stop" demands, poor Go performance and higher NoGo performance could be driven by inattention to that cue. Future research may seek to solve this problem using tasks such as the stop-signal which have separate cues for Go and Stop, so Go performance is unambiguously about attention (and responses) to only the Go cue, and Stop performance is unambiguously about the ability to inhibit an established prepotent response.

The finding that higher maternal parenting stress was associated with children's greater attentional impairment following acute stress suggests that individual differences in children's vulnerability to stress may be linked to intergenerational influences. We posit that young children who have parents that are less able to model effective coping strategies to acute stress or to provide social buffering in the form of sensitive responding (Hostinar et al., 2014; Propper & Holochwost, 2013) have reduced capacity to recover from acute stress and effectively engage in challenging cognitive tasks. However, other interpretations are also plausible, including a potential for heritable influences; parents who have difficulty managing stress may be more likely to have children with difficulty managing stress. The high collinearity between subscales of the PSI ($r = .54-.62$) makes a fine-grained discrimination of specific parenting processes relevant to children's coping challenging. Future research should employ more focused assessments, such as observed maternal sensitivity or emotional modeling, to understand how these factors influence children's coping abilities. Future research may also seek to employ more rigorous assessments of proximal environmental influences on children's cognitive vulnerability following acute stress. For example, it would be valuable to determine the extent to which caregivers' own autonomic reactivity and cognitive function following acute stress influence children's self-regulatory function in addition to delineating if caregiver's presence during, or immediately after, acute stress could support children's recovery and buffer them from subsequent attentional impairment.

A major gap in this literature is a clear understanding of the processes by which a series of acute stress exposures accumulate to cause the effects associated with chronic stress. The data presented here only begin to address that gap. In Figure 4, we formulate an

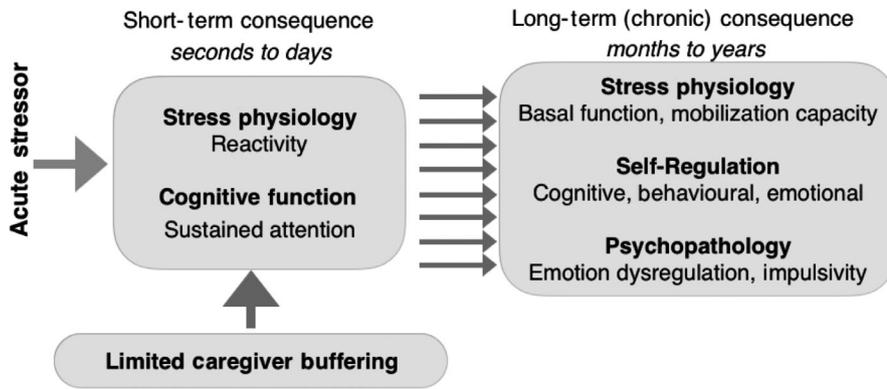


FIGURE 4 Conceptual model of candidate pathways through which repetitive exposure to acute stress may contribute to long-term difficulties in children's self-regulatory function

initial conceptual model that illustrates candidate pathways through which repetitive exposure to acute stress and the resulting short-term cognitive and physiological consequences may contribute to long-term difficulties in children's self-regulatory function. In particular, it is theorized that, over time, the repetitive experience of altered sustained attention following acute stress may cause trait-level impairment in children's ability to effectively process environmental input and develop the top-down attentional and behavioral regulatory skills necessary for goal-directed behavior. Notably, this model builds on social buffering theory in early childhood (Gunnar et al., 2015; Hostinar et al., 2014) to suggest that greater caregiver capacity to buffer children from the effects of acute stress, through processes such as emotional support and modeling of effective stress management strategies, contributes to reduced short-term attentional impairment due to acute stress, in addition to dampening the reactivity of children's stress response systems. Future longitudinal research should investigate links between acute stress vulnerabilities and long-term impairments in self-regulation and psychosocial function.

There was no evidence for alterations in children's SNS and PNS acute stress reactivity underlying cognitive vulnerability to acute stress in the present sample. Notably, this was an exploratory area of research, given the minimal prior research in this area. One prior adult study documented that RSA reactivity to an acute social stressor was linked to post-stressor inhibitory control impairments (i.e., stop signal reaction time; Roos, Knight, et al., 2017). The PNS may be more relevant to buffering children from the effects of acute stress for cognitive skills that require more effortful regulation of motor responses, such as inhibitory control, and less-so for sustained attention given the critical role of shared brain regions in modulating both inhibitory control and PNS regulation (Thayer et al., 2012). Another possible explanation for the lack of association between PNS activity and performance accuracy reported here is that the high-frequency bandwidth used here (0.24 to 1.04 Hz) may have underestimated PNS levels in this sample (Shader et al., 2018). It was also interesting that SNS function was not associated with behavioral impairment given that higher parent distress predicted increased SNS engagement (i.e., shortening of PEP) to the stressor, an effect which is consistent with previous research documenting heightened SNS reactivity in children of mothers with high distress

(Monti, Abaied, & Rudolph, 2014). It is possible that we were underpowered to detect links between SNS and PNS function and children's cognitive impairment following acute stress; however, establishing effect sizes was difficult given the lack of prior research in this area. Future research may also benefit from more dynamic approaches to examining SNS and PNS reactivity, such as approaches that examine growth models over the course of a task (e.g. Miller, Nuselovici, & Hastings, 2016). When considering such an approach for the acute stressor used here, it will be important for researchers to consider standardizing the onset and offset of each of the three blocks of the matching task employed during the stressor.

In future work, it will be valuable to replicate and extend the present findings to samples that include children with clinically significant externalizing problems for whom the effects of acute stress on self-regulatory process might be exaggerated. Similarly, examining the role of the acute stressor on cognitive performance would be particularly interesting in samples with exposure to high levels of chronic stress in early childhood (e.g., due to maltreatment or maternal depression). Longitudinal research is also needed to examine if children who exhibit impaired post-stressor performance are the same children who may come to develop deficits in cognitive function over time. This would provide an important candidate mechanism for helping to explain links between cognitive function with both chronic stress and parenting behavior (Cowell et al., 2015; Fay-Stammach et al., 2014; Pechtel & Pizzagalli, 2011).

Prior research examining the effects of early life stressors, including institutionalized neglect and severe abuse, have highlighted the effects of these experiences on children's impairments in sustained attention and not inhibitory control (Lim et al., 2016; Loman et al., 2013; Xue, Lin, Sun, & Cao, 2017). Although early life chronic stress is certainly associated with negative developmental outcomes linked to impulsivity, such as substance use and risk-taking, it is possible that these outcomes have a different etiology based on earlier-stage cognitive processes. In a recent review, we have argued for the value of employing translational neuroscience approaches for informing questions about the extent to which the timing of chronic stress exposures differentially affects brain development based on sensitive periods and the importance of such differences in informing intervention and prevention efforts early in life (Roos, Horn, Berkman, Pears, & Fisher, 2019).

Taken together, the results presented here provide the first evidence that acute stress impairs cognitive performance in young childhood. Given that young childhood represents a period of vulnerability to stress during which children are gaining self-regulatory autonomy and increased independence, a more refined understanding of acute stress effects on cognitive performance during this time has particular significance. Indeed, children in this age range are asked to perform many cognitively demanding tasks and encounter potentially stressful situations such as meeting academic and social expectations in new environments (e.g., school). Impairment following acute stress may be particularly detrimental in this context and contribute to ongoing adjustment challenges in young childhood and beyond. Notably, parenting stress was predictive of acute stressor-related declines in sustained attention, consistent with previous theory about the critical nature of the mother-child relationship for child regulation in response to acute stress (Hostinar et al., 2014). Identifying parenting stress as a risk factor for vulnerability to the effects of acute stress has particular translational relevance for interventions because it may offer a proximal target through which to increase children's resiliency during young childhood. Indeed, there is growing evidence that maternal (or caregiver) stress may be malleable through intervention (e.g., parent management training, mindfulness training, and dialectical behavior therapy; Fisher & Stoolmiller, 2008; Martin, Roos, Zalewski, & Cummins, 2016; Neece, 2014). Future research using comprehensive and multi-modal measures of caregiver stress management and the parent-child relationship will be important for further elucidating how parenting stress impacts children's cognitive vulnerability to acute stress and may be targeted via intervention. For example, parenting stress may contribute to acute stress vulnerability through diminished overall maternal sensitivity contributing to poorer child attachment or lower internalization of effective stress-regulation strategies (Gunnar, 2017). This would suggest that any program that can improve maternal sensitivity as a global construct should also improve children's resilience to acute stress. Alternatively, it may be that mothers with high parenting stress are in particular need of skills to support children in coping with and recovering from distressing or challenging stressful events, which would suggest the need for a more targeted intervention specific to the stressor context. Gaining additional understanding of environmental and biological factors that increase children's vulnerability to acute stress may allow for future interventions to bolster children's regulatory capacities in the face of acute stress and ameliorate the effects of more chronic stressors on cognitive development.

CONFLICT OF INTEREST

The authors have no conflicts of interest to report.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on the open science framework at <https://osf.io/7asqp/>.

ORCID

Leslie E. Roos  <https://orcid.org/0000-0001-7083-4017>

Elliot T. Berkman  <https://orcid.org/0000-0002-7113-5792>

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How to cite this article: Roos LE, Giuliano RJ, Beauchamp KG, Berkman ET, Knight EL, Fisher PA. Acute stress impairs children's sustained attention with increased vulnerability for children of mothers reporting higher parenting stress. *Developmental Psychobiology*. 2020;62:532–543. <https://doi.org/10.1002/dev.21915>